

Definition

Tinnitus is the sensation of a noise in the ear or head when no apparent source for the noise is evident. Tinnitus may be either subjective (perceived only by the patient) or objective (perceived by an examiner also). Virtually 95 to 98% of tinnitus is subjective, and 65 to 98% of tinnitus is idiopathic. Unfortunately, tinnitus is one of the most common disorders known. The American Tinnitus Association reports that over 20 million Americans have this distressing symptom.

Technique

To identify an associated disorder the history is exceedingly important, because certain reversible problems (e.g., recurrent exposure to loud noise), drugs producing tinnitus (e.g., aspirin or quinidine), barotrauma from scuba diving, renal disease with ototoxic drug exposure, or lymphoma treatment involving ototoxic drugs may be discovered.

The examiner must quickly run through a list of various groupings of disorders. These include otologic, dental and orthodontic, traumatic, metabolic, neurologic, pharmacologic, or psychiatric (Table 121.1).

The patient should be questioned as to duration, intensity, quality, pitch, pulsatile nature, fluctuation, sleep effects, and resolution of the noise. A determination should be made as to exact location. Is the sound in one or both ears, elsewhere in the head, or does it extend into the neck, eye, face, or temple?

Careful review of the history may indicate excessive noise exposure, head injury, or exposure to ototoxic drugs. In less than 40% of cases, however, can any definite association be noted in subjective tinnitus.

A throbbing sensation in the ear or head may indicate objective tinnitus. The patient may mention that lying supine, rotating the head, or pressing on the neck will either exacerbate or eliminate the noise. Other persons can hear an objective tinnitus either by holding the ear close to the patient's head or with a double stethoscope. Sometimes a correctable cause for objective tinnitus, such as a glomus tumor or aneurysm, can be found.

Only rarely, however, can a cause amenable to medical or surgical treatment be identified in subjective tinnitus cases.

Basic Science

Some authorities believe that tinnitus represents the loss of a damper mechanism that all human beings possess normally. This mechanism may be operative through the bundle of Rasmussen, which is a set of efferent fibers running from the reticular formation in the brainstem to final endings on the hair cells of the cochlea. Just as parkinsonism

represents a loss of control by the basal ganglia, perhaps tinnitus represents loss of eighth nerve suppression by some areas of the brainstem or midbrain.

Patients do not die of tinnitus, and very little clinicopathological information exists. The ubiquitous nature of the disorder indicates a vital need for long-term prospective studies for both premortem and postmortem documentation of pathological changes.

A comprehensive study of patients with tinnitus was carried out by the Kresge Hearing Research Laboratory and the Tinnitus Clinic of the University of Oregon Health Sciences Center. The age distribution was: 58% age 45 to 65; 18% age 65 to 80; 11% age 20 to 35. The oldest patient was 85, the youngest, 14. The very young seemed to have congenital problems. The older patients had mostly noise-induced tinnitus. The sex ratio was 65% male and 35% female. Some patients in the study had experienced the tinnitus unilaterally, but the majority (58%) had it bilaterally.

Normally the two ears have tinnitus of the same pitch, but often the loudness is unequal. It is rare to find the pitch in one ear differing from that in the other. The loudness may vary between ears. Only 5% of patients localized the

Table 121.1
Causes of Tinnitus

Otologic

Ear infections, fluid, trauma, tumors or previous surgery, impacted cerumen, idiopathic ear disorders such as otosclerosis or Ménière's disease, eustachian tube malfunction—often allergic or related to birth control pills

Metabolic

Lipoprotein or blood pressure disorders

Dental and orthodontic

History of braces, root canal therapy, recurrent dental work requiring the mouth open for 30 to 60 minutes, poor-fitting dentures

Traumatic

Jaw, head, and neck injury; barotrauma

Neurologic

Acoustic neuroma, cerebellopontine angle tumor, basilar aneurysm, previous meningitis, multiple sclerosis

Pharmacologic

Salicylates, aminoglycoside antibiotics, furosemide and ethacrinic acid, quinine and derivatives, antiarthritic drugs, methotrexate or other chemotherapeutic drugs

Psychiatric

Schizophrenic patients often complain of sounds intracranially, poorly localized and varying in quality and intensity. The associated disorders listed above are missing. Hearing is normal and often supernormal with hyperacusis. Neurotic patients have marked accentuation of an underlying mild tinnitus during times of anxiety or depression.

tinnitus within the head. The localization in the head may be due to equal presentations from each ear; it may also be due to head injury.

Severity of the tinnitus does not correlate with the loudness as reported by the patient. Most patients describe the loudness of their tinnitus as 5 to 10 dB. This is very low, considering that normal conversation is 55 to 60 dB. Severity seems related to some factor other than loudness.

Some patients have fluctuating tinnitus that may actually disappear at times. This cannot be correlated to any set pattern. In 68% of the University of Oregon patients the tinnitus was constant; in 32%, fluctuating.

The pitch of the tinnitus seems to fit two broad categories: tonal tinnitus and noise tinnitus. The pitch of the tonal type can usually be categorized, but the noise type cannot. In 59% of patients tonal tinnitus was present; 25% had noise-type tinnitus; 16% seemed to have a combination. The patients were classified as 63% between 2000 and 7000 Hz. Only 21% had low-tone tinnitus below 2000 Hz and 16% above 7000 Hz. This tinnitus is high pitched.

In most individual cases it is impossible to determine the cause of tinnitus. Head injury was identified in 35% of the University of Oregon cases. The tinnitus of head injury seems to fluctuate widely. Residual inhibition (absence of sound) may occur, which allows freedom from the tinnitus for a period of time.

All of the above comments refer to patients with idiopathic, subjective tinnitus. The natural history of objective tinnitus follows the course of the tumor, aneurysm, or vascular abnormality that caused the noise.

Clinical Significance

In identifying causes amenable to treatment, most tinnitus (85%) will end up being idiopathic. Perhaps 5% will be

objective, and the other 10% will demonstrate one of the possible causes listed in Table 121.1. Effective treatment may be possible against objective types such as glomus jugulare tumors, anomalous middle ear vessels, or aneurysms or vascular anomalies in and around the temporal bone. Otologic consultation with complete audiometry is needed in all patients with chronic tinnitus (6 months' duration or more). In tinnitus of shorter duration and sudden onset, some obvious related disorder, such as viral infection or noise exposure, can usually be ascertained.

Virtually all cases of sudden tinnitus are associated with other symptoms, such as hearing loss, pain, headache, fullness in the ear, vertigo, nasal congestion, otorrhea, nystagmus, or hyperacusis. The reader is referred to topics dealing with the management of these related symptoms. Tinnitus is seldom a solitary symptom.

References

- Jackson RT, Todd NW, Burson JH, Turner JS. Drug induced ear, nose and throat disease. In: Drug treatment, 3rd ed. Baltimore: Adis Press, 1987;380.
- Lubin MF. Tinnitus. In: Walker HK, Hall WD, Hurst JW, eds. Clinical methods, 2d ed. Woburn, MA: Butterworths, 1980;71-76.
- Vernon JA. Tinnitus. In: ATA Newsletter. Portland, OR: American Tinnitus Association, 1978;3.